CASE REPORTS

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Surgical Management of Gastroparesis Diabeticorum

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GASTROPARESIS DIABETICORUM was described by Kassander[1] as gastric atony seen in diabetes mellitus. Twenty-one patients with this syndrome have been presented in the literature. All but two of them were treated conservatively, with varying success. Most of the patients showed no significant improvement. Wooten[2] presented two cases treated surgically with only partial symptomatic relief. Wooten[2] and Howland[3] said that surgical operation may be contraindicated. Our experience includes two cases in which surgical management was effective.

Reports of Cases

Case 1. The patient, a 44-year-old former nurse, was first seen at Stanford University Hospital on May 6, 1965, for nausea, vomiting and weight loss of four months duration. She had had known diabetes for seven years and had been in good health until the onset of that disease, which was in good control on 40 to 50 units of insulin per day and a 2000 calorie diet.

In January, 1965, the patient changed physicians and an attempt was made to control her diabetes with chlorpropamide (Diabinese®). The disease went out of control, and nausea and vomiting developed, for which she was admitted to hospital and treated with methantheline (Banthine®). Later a streptococcal throat infection developed and she became febrile, dehydrated and acidotic. A 15-pound weight loss ensued. Neuritis developed, with causalgia in the lower back, and urinary hesitancy. Four months before admission to Stanford, insulin injections were resumed. She began to vomit occasionally and was treated with propantheline (Pro-banthine®) with only minimal relief. An upper gastrointestinal series suggested pylorospasm. Urecholine was tried, but because of diarrhea it was discontinued. The patient then was admitted to Stanford University Hospital for further evaluation and treatment. She was thin (signs of recent weight loss) with dry skin and facial hirsutism. There were no microaneurysms in the fundi. The chest was clear to percussion and auscultation; the pulse rate was 110 and blood pressure was 110/68 mm of mercury. Heart sounds were within normal limits. The abdomen was soft and bowel sounds were present. Deep tendon reflexes were depressed and the vibration and position sense was poor.

The urine had a 1 plus reaction for glucose, a trace of acetone, 1 to 3 leukocytes per field, no erythrocytes, no bacteria. The blood contained 10,600 leukocytes per cu mm with 62 percent neutrophiles, 19 percent lymphocytes, 9 percent monocytes, 9 percent eosinophiles, 1 percent basophiles and a few macrocytes. The packed cell volume was 37.5 percent and the hemoglobin content 11.6 grams per 100 ml. Electrolytes were all within normal range. Glucose at that time was 348 mg per 100 ml. In 12-hour gastric aspirate volume of 1.15 liters there were 1.8 units of free hydrochloric acid and total acid of 60 units. Serum magnesium was 1.5 mg per liter (normal range 1.5 to 2.4 mg). Histamine-stimulated gastric secretion on June 3, 1965, showed a

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basal secretion of 13.4 mEq and stimulated secretion of 27.4 mEq of hydrochloric acid. D-xylose excretion was 1.4 grams in 5 hours (less than normal range). Fecal fat was 5.38 grams per 24 hours while the patient was on no fat intake. This value increased to 16.9 grams of fat excreted on a 70 gram fat intake. Examination of stool for ova and parasites was negative on three examinations.

X-ray examination of the upper gastrointestinal tract and small bowel on June 14, 1965, showed relative obstruction at the pylorus (2mm), thought to be functional, as no organic lesions were seen. A motor meal was retained in the stomach ten hours. X-ray films of the lumbosacral spine and knees showed moderate osteoporosis. (Serum calcium at that time was 8.3 mg per 100 ml). The patient was originally treated with suction drainage of the stomach. During the admission period her diabetes was difficult to control and the blood sugar content ranged from 65 to 465 mg per 100 ml. She had several incidents of acetonemia and acetonuria. Nausea, vomiting and diarrhea persisted. It was felt that the diabetes was improperly controlled and that peripheral and autonomic neuropathies were the cause of symptoms. A succussion splash could still be heard and there were several incidents of emesis. Urecholine was tried again and caused diaphoresis and emesis. The patient became depressed over the long stay in hospital and the difficulty in ameliorating her symptoms.

Peritoneoscopy was done June 8, 1965, and no organic lesion was seen. On June 15, six months after onset of symptoms, pyloroplasty was carried out. The postoperative course was good but diarrhea continued—at least five stools a day. Fecal fat studies confirmed the diagnosis of malabsorption. Symptoms slowly regressed and on July 12, 1965, an upper gastrointestinal study showed transit time of approximately two hours with no evidence of obstruction at the pylorus. Three post-operative 12-hour gastric aspirates showed volumes of 365 to 425 ml, with free acids ranging from 20 to 33 units and total acids 40 to 56 units.

The patient was released July 13, 1965, and she had no further problems relative to gastric retention.

Case 2. The patient, a 24-year-old white woman had a 12-year history of diabetes mellitus in fairly good control, with occasional incidents of coma, but not ketoacidosis. She was maintained on 15 units of regular insulin and 45 units of lente insulin a day. Five months before her first Stanford University Hospital admission, the patient began a diet to reduce from her regular weight of 180 pounds. A month later, nausea and vomiting developed and she was admitted to another hospital and treated with tranquilizers. Her condition was somewhat improved and remained so until, two months before admission to Stanford, nausea and vomiting recurred and acetonuria developed. She was admitted to another hospital for one week but no cause for the gastrointestinal symptoms was discovered. Two weeks before admission to Stanford, nausea and vomiting recurred and the patient was again admitted to a different hospital. Results of liver function tests and of pancreatic screens were all shown to be within normal limits. Pregnancy tests were negative. An upper gastrointestinal series at that time showed an enlarged stomach with delayed gastric emptying. The patient was transferred to Stanford University Hospital on May 23, 1968, for evaluation and treatment of gastric atony.

At the time of admission the patient was taking 35 units of lente insulin. She complained of nausea and vomiting, diarrhea and epigastric pain after meals and had lost 35 pounds in four months. She had been having acetonuria and glucosuria for a few weeks before admission.

On physical examination she was observed to be moderately obese and in no acute distress. Ophthalmoscopic examination revealed evidence of arteriolar narrowing. Several microaneurysms with small hemorrhages were seen. The chest was clear to auscultation and the heart size was within normal limits. Tenderness and distension were noted on epigastric palpation. Neurological examination showed no deficits. It was felt by a psychiatric consultant that the patient had a neurotic component to her personality. Laboratory data were within normal limits. Blood sugar ranged from 110 to 303 during the period of admission. The urine gave a 4 plus reaction for glucose and 3 plus for acetone. Plasma glucose was 174 mg per 100 ml on admission. Gastric analysis showed no free acid in a 13-hour collection. An augmented histamine test revealed a basal acid secretion of 0.7 mEq, which rose to 13.0 mEq with histamine.

An upper gasrointestinal tract study showed delayed gastric emptying, and generalized slow

peristalsis was observed fluoroscopically. A motor meal study showed that the stomach took 12 hours to empty completely.

An electrogastrogram showed normal waves with a slowed rate. After four months of symptoms, Heineke-Mikulicz pyloroplasty was performed. No organic lesions were found. The patient tolerated the procedure well and the symptoms were relieved. A postoperative 12-hour gastric aspirate contained 1.2 mEq of free acid. The patient was discharged a week after operation, asymptomatic. She was taking 35 units of lente insulin and the blood sugar was 200 mg per 100 ml.

On follow-up in our clinic, an upper gastrointestinal series two and a half years after operation revealed no evidence of gastric distention and a histalog test showed a basal secretion of 0.1 mEq and a test response of 15.2 mEq. The patient was still asymptomatic.

Discussion

Gastroparesis diabeticorum as described by Kassander[1] is "asymptomatic" although some of the patients he presented in his paper complained of epigastric fullness and anorexia. These complaints were not diagnostic and the final diagnosis was made from radiologic evidence. It is interesting that 15 of the 21 patients presented in the literature had vague symptoms referable to gastric retention.

Radiologic diagnosis of this entity was discussed by Marshak[2] and Gould[3]. The stomach appears grossly distended, with loss of the rugal pattern. This might be interpreted as pyloric stenosis except that the barium can be manually expressed from the stomach, showing that the gastric distension is not due to mechanical outlet obstruction. The transit time of barium is decidedly prolonged (up to 24 hours). Motility of the stomach was decreased in approximately a fourth of these patients and the amount of stomach secretions was greater than normal. These changes are reminiscent of those that occur after vagotomy. Kravetz[4] observed similar findings at gastroscopy.

Many observers have conjectured upon the pathogenesis of this syndrome. There is a form of gastric atony associated with ketoacidosis but this syndrome is not associated with acidosis. Thus, acute metabolic imbalance cannot be construed to be the cause of this problem.

Dotevall[5,6] in a study of gastric motility in diabetic patients showed that those with advanced disease had delayed gastric emptying when given saline test meals. Angervall[7] observed angiopathic change in the gastric mucosa of patients in whom delayed emptying had been demonstrated. Rundles[8] in an article on diabetic neuropathy described some diabetic gastrointestinal disturbances as being due to the neuropathic changes in the visceral nerves. Ellenberg[9] agreed that gastric distension and diabetic diarrhea were both due to visceral neuropathy. Dotevall said that diabetic neuropathic changes of the vagus nerve could account for all the components of gastroparesis. From these observations it would appear that the syndrome is very close to the post-vagotomy state.

Nelsen[10] et al in a study of the motor and electrical functions of the stomach after vagotomy, showed that there is slowing of the electrical and mechanical rates. A desynchronization analogous to atrial fibrillation is present, in which there is ineffective ortho grade flow. Beck and Mason[11] showed that a rapid focus of contraction is present in the pyloric antrum and postulated that post-vagotomy stasis may be related to antiperistalsis from this focus. With these experimental models, one can more easily understand how gastric stasis may result from vagal neuropathy.

Conservative treatment for this condition has been recommended in the literature. Howland[12] wrote that conservative therapy is the only valid treatment, and expressed belief that surgical operation is contraindicated. Conservative management consisted of strict diabetic control and a six equal meal diet. Many patients did not respond to this therapy and their symptoms persisted. Wooten[13] reported two cases in which surgical management was tried. One patient had partial gastrectomy and Billroth I anastomosis because of suspected ulcer disease. He began to gain weight on a six feeding diet after operation. The other patient had pyloroplasty. Although this patient had persistent nausea and vomiting after the operation, epigastric pain was relieved.

The two patients presented here both had pyloroplasty with good results after failure of medical therapy. Follow-up has shown no adverse effects of the operative procedure. Both had significant improvement of the symptoms related to gastroparesis. Neither had a significant alteration in gastric secretion postoperatively. As a result

of this experience we believe that pyloroplasty may be of benefit when conservative measures fail in patients who present with the gastroparetic type of diabetic gastroenteropathy.

Summary

The treatment of gastroparesis diabeticorum recommended in the literature has always been conservative management. Two patients presented herein were successfully treated with pyloroplasty.

This condition is not easily diagnosed on clinical grounds. The radiographic and gastroscopic findings are characteristic. The most likely pathogenesis is related to a diabetic vagal neuropathy.

Pathophysiologically this condition is reminiscent of the post-vagotomy state. The inefficient orthograde propulsion of gastric contents may be facilitated by a surgical drainage procedure, such as pyloroplasty. Surgical management may therefore be considered an alternative to conservative management of diabetic gastric atony.

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Symptomatic Lower Esophageal Rings—Treated Endoscopically

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IN A RECENT REVIEW, Goyal et al,[1] summarized results of a variety of therapeutic measures in patients with symptomatic lower esophageal rings. These rings are usually of the mucosal type and frequently are associated with hiatus hernia. Intermittent dysphagia is generally one of the main symptoms. The symptom complex has been called the "steakhouse syndrome"[2] because often the initial symptoms occur when the swallowing of a large bolus of meat causes temporary obstruction. Delmonico[3] in 1956 first reported successful treatment of a symptomatic ring by forceful rupture with an esophagoscope. Subsequently, Somm et al,[4] using an esophagoscope with external diameter equivalent to a no. 16 bougie, severed one portion of the ring with a punch forceps to relieve symptoms. Norton and King[2] and Postlethwaite and Sealy[6] used Hurst bougienage with good results. Adams[7] and Mossberg,[8] Hyatt[9] and Riegel[10] reported good results with pneumatic dilators. Goyal prefers the technique of Hurst bougienage, using first a no. 16 to no. 18 bougie, and following it immediately with a no. 48 without intervening sizes. He believed that procedure preferable to esophagoscopic rupture.

In the past few years I have treated three patients with symptomatic lower esophageal rings by esophagoscopic rupture of the ring.

Reports of Cases

Case 1. A 48-year-old white woman was seen in October 1965 with a history of intermittent dysphagia over a five-year period. Two and a half years previously, gastrointestinal x-ray stud-

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